

1 MICHAEL J. O'TOOLE (SBN 97779)

City Attorney

2 RANDOLPH S. HOM (SBN 152833)

Assistant City Attorney

3 CITY OF HAYWARD

777 "B" Street

4 Hayward, California 94541

Telephone: (510) 583-4450

5 Facsimile: (510) 583-3660

6 Attorneys for Defendants City of Hayward, Craig

Calhoun, R.Farro, A.Nguyen, E. Mulhern,

7 C. Martinez, E. Hutchinson, J.Waybright,

R. Sappington, D.Olsen, erroneously

8 sued herein as D. Olson, J. Bryan, and R.Keener

9 UNITED STATES DISTRICT COURT

10 NORTHERN DISTRICT OF CALIFORNIA

11 ANNIE LEWIS, et al.,

Case No.: C 03 -5360 CW

12 Plaintiffs,

DECLARATION OF TOM NEUMAN,
M.D.

13 -vs-

14 CITY OF HAYWARD, et al.,

15 Defendants.

16 _____/
17 I, TOM NEUMAN, M.D., declare that:

18 1. I am a physician at the Department of Emergency Medicine, University of
19 California, San Diego (UCSD) Medical Center, 200 W. Arbor Drive, San Diego, California
20 92103. I am a physician and Professor Medicine and Surgery at the UCSD School of Medicine. I
21 have over thirty years experience as a physician practicing Emergency Medicine, Pulmonary
22 Medicine and Undersea and Hyperbaric Medicine. During the course of my career, among other
23 things, I served on the San Diego Coroner's Committee for Investigation of Diving Fatalities, the
24 City Manager's Task Force on Carbon Monoxide Poisoning, as a NASA consultant, and as a
25 Captain in the U.S. Naval Reserves. I am separately board certified in the disciplines of Internal
26 Medicine, Pulmonary Disease, Occupational Medicine, Emergency Medicine, and Undersea and
27 Hyperbaric Medicine. I am a diplomate of the National Board of Medical Examiners, and I am
28 licensed to practice medicine in the State of California. I hold a bachelors degree from Cornell

1 University, and received my medical degree from New York University School of Medicine. I
2 was retained by the Defendants in the above case to provide my expert opinions regarding the
3 incident involving the Hayward Police Department (“HPD”) and the decedent, Gregory Tyrone
4 Lewis out of which this legal action arises, including but not limited to the cause of death, and
5 plaintiffs’ allegations regarding restraint asphyxiation. Attached hereto is a true and correct copy
6 of my Federal Rules of Civil Procedure (“FRCP”) 26 Report that I prepared in relation to the
7 within action, which includes a true and correct copy of my resume.

8 2. In the case at hand, I have had the opportunity to review the items identified in my
9 Rule 26 Report including but not limited to the autopsy reports prepared by Tom Rogers, M.D.,
10 and Plaintiffs’ forensic pathologist, John T. Cooper, M.D [both of which indicate the level of
11 drugs found in Mr. Lewis, and the lack of a physical injury sufficient to cause his death (i.e.
12 “negative pathology”)], the Hayward Police Department (“HPD”) Incident Report No. 2002-
13 03598, Mr. Lewis’ medical records including his records from St. Rose Hospital and Fairmont
14 Hospital (indicating that Mr. Lewis was a “substance abuser - smokes crack, etc.”), his criminal
15 history indicating drug related crimes over the past several years, the toxicology results prepared
16 by Nikolas Lemos, PhD., Laboratory Director and Chief Forensic Toxicologist at the Office of
17 the Chief Medical Examiner for the City and County of San Francisco and the deposition
18 transcripts and/or declarations of the percipient witnesses to the incident (describing Mr. Lewis’
19 violent physical resistance and bizarre behavior) listed in my report.

20 3. Based on my training and experience, it is my expert opinion that Mr. Lewis died
21 of the combination of his use of PCP and cocaine as well as his underlying heart disease, which
22 resulted in lethal arrhythmia. From the perspective of an Emergency physician, it appeared Mr.
23 Lewis was suffering from “excited delirium” which in turn was probably secondary to his drug
24 use.

25 4. Persons with excited delirium usually display violent behavior to the police
26 officers who are summoned. Since these individuals usually do not respond to or follow the
27 instructions of the officers, the officers are then compelled to resort to chemical agents such as
28 pepper spray, or physical restraints to control the subject. At present, in large inner city

1 emergency departments, most people presenting with the symptoms of excited delirium are not
2 individuals with intrinsic mental disease but rather individuals whose symptoms are due to illicit
3 stimulants. Importantly, under many circumstances, the officers cannot differentiate between
4 symptoms that are due to mental disease and the symptoms that are due to illicit stimulants. In
5 fact, such differentiation may not be possible even by medical personnel unless a patient's history
6 and/or toxicology testing are available.

7 5. To better understand Mr. Lewis' death one must also understand the historical
8 background of "excited delirium" and the pseudo scientific origin of the theory of positional
9 asphyxia secondary to restraint. In 1981, D.A. Fishbain, M.D. and C.V. Wetli, M.D. published an
10 article in the Annals of Emergency Medicine ⁽¹⁾ that reintroduced the concept of death in
11 association with excited delirium. The article focused on cocaine intoxication and the death of a
12 body packer (person that smuggles illegal narcotics like cocaine by swallowing packets of the
13 drug in plastic bags, balloons, or condoms or by inserting such packets into the rectum).

14 6. In 1985, Wetli and Fishbain reported on seven deaths associated with excited
15 delirium in an article titled Cocaine-Induced Psychosis and Sudden Death in Recreational
16 Cocaine Users.⁽²⁾ Of the seven cases presented, all presented in excited delirium and all were
17 restrained. Five died in police custody and two in medical custody. In none of the seven cases
18 were neck holds used and none were placed in situations where mechanical asphyxia was
19 possible. In this article, the authors found that the use of restraints did not contribute or cause
20 death.

21 7. However, in 1988, D. T. Reay, M.D. and his colleagues⁽³⁾ conducted a single
22 experiment to determine the effects on peripheral oxygen saturation and heart rate that occur after
23 an individual is hog-tied and placed prone following exercise. Peripheral oxygen saturation and
24 heart rate were determined using a pulse oximeter. Reay and his co-workers concluded that hog-
25 tie restraint prolongs recovery from exercise due to an "alleged" increase in time to return to
26 "normal" of peripheral oxygen saturation and heart rate in subjects placed in the hogtie position.
27 They speculated (and I emphasize that "speculated" is the correct word as in their experiment
28 they made no measurements of ventilation whatsoever) that restriction of thoracic respiratory

1 movement could be one of the mechanisms for this occurrence and recommended that positional
2 restraint and its effects should be considered in the investigation of individuals restrained in the
3 prone position. They did not consider the possibility that their results were erroneous nor did
4 they examine their methodology for possible flaws. In the last sentence of that paper they
5 recommended, “additional research is needed to better understand the pathophysiology involved
6 in these deaths” ⁽³⁾. “Additional research” was ultimately done by my co-workers and I in 1997.⁽⁴⁾
7 Unfortunately, in the interim period, Dr. Reay’s 1988 paper served as the basis for the diagnosis
8 of positional asphyxia from restraint by him as well as other physicians including but not limited
9 to Ron O’Halloran, M.D. Furthermore this paper “led to misconceptions regarding the
10 physiological results of the use of restraints and numerous lawsuits” ⁽⁵⁾.

11 8. In 1992, Reay published a paper titled “Positional Asphyxia during Law
12 Enforcement Transport.” ⁽⁶⁾ He described three cases in which individuals died after being hog-
13 tied and placed prone in the rear of police vehicles. One individual died while being transported
14 to a hospital and the other two died while they were being transported to jail. Two of the three
15 had a history of endogenous mental disease and one was under the influence of alcohol,
16 marijuana, and LSD. Reay’s and his co-workers attributed the three deaths to positional asphyxia.
17 Reay’s paper indicated “positional asphyxia occurs when the position of the body interferes with
18 respiration resulting in asphyxia.” but once again no measurements of any sort were made to
19 support these conclusions and therefore this hypothesis must be viewed as no more than
20 speculation.

21 9. In 1993, in an article by R.L. O’Halloran and L.V. Lewman⁽⁷⁾, the authors
22 associated restraint, asphyxiation, hogtying, and death into the concepts of “restraint asphyxia” or
23 “positional asphyxiation”. Importantly, this paper relied upon Reay’s unsubstantiated (and as I
24 will explain later, scientifically flawed) restraint asphyxiation theory (page 295, reference
25 number 6 of that paper). These authors reported eleven cases of individuals in excited delirium
26 who died following restraint in a prone position: nine were hog-tied, one tied to a hospital
27 gurney, and one held prone manually; of the individuals, six were under the influence of cocaine,
28 one methamphetamine, one LSD, and three had intrinsic mental disease. Two of the hog-tied

1 individuals died in the back of police vehicles. All individuals presented with excited delirium
 2 with the symptoms less than one to six hours in duration. All required several individuals to
 3 control and restrain them; all were restrained prone; all continued to struggle when restrained.
 4 The authors concluded, “the mechanism of death appears to be a sudden cardiac dysrhythmia or
 5 respiratory arrest induced by a combination of at least three possible factors relating to increased
 6 oxygen demands and decreased oxygen delivery”. According to them, these factors were:

- 7 a. Stress on the heart due to catecholamine release from the excited delirium
- 8 b. The hyperactivity of excited delirium; the resultant struggle with police and/or
 9 medical personnel and struggling against the restraints increased oxygen demand
- 10 c. Hog-tying impaired breathing by inhibiting chest wall and diaphragmatic
 11 movement in face of the increased oxygen demands

12 Once again, and as in all of the case series reporting deaths taking place in people who
 13 were restrained, no measurements whatsoever of ventilatory function were made to support these
 14 speculations. Significantly, since the foundation of this paper was based upon the work of Reay,
 15 et al. which was later disproved by our studies, (which were performed to examine the effects of
 16 pepper spray, prone position, hogtie (maximal restraint), and weight force placed on a suspect)
 17 ^(8,9,10,11,12) and which was recognized to be fatally flawed in Price v. County of San Diego, 990
 18 F.Supp. 1230 (S.D. Cal. 1998), the conclusions by O’Halloran are likewise flawed.

19 10. In 1995, Stratton et al., ⁽¹³⁾ described two cases of sudden death associated with
 20 excited delirium and restraint during a transport by medical personnel. This paper remains
 21 important because the individuals in this study were being monitored when they died and
 22 resuscitation was begun immediately. Both individuals were hog-tied. The first individual was a
 23 35-year-old male on methamphetamine who was transported while hog-tied in a prone position.
 24 His heart rate went from 136 to 60 beats/minute, and then went up to 102, and then to asystole,
 25 all within a minute. The other individual was under the influence of cocaine and
 26 methamphetamine and suffered a rapid asystolic arrest. Despite the fact that the arrests were
 27 observed and despite the immediate institution of resuscitation by trained and equipped
 28 Advanced Life Support (ALS) personnel, both patients died.

11. The concept that deaths of individuals who are restrained (as is almost universally required in cases of “excited delirium”) are due to positional or restraint asphyxia was examined objectively and appropriately for the first time in our laboratory in 1997. In order to understand our work, it is also necessary to understand the basic flaws in Dr. Reay’s previous work. Basically, his premise was incorrect, the methodology flawed, the statistics were incorrectly applied, and the synthesis illogical. Specifically:

a. The author’s premise for the experiment depended upon the assumption that a drop in oxygen saturation occurred with exercise. Unfortunately basic physiology textbooks show improvement in arterial oxygenation with moderate exercise^(14,15,16). As Dr. Reay testified (Price v. San Diego) he was unaware of this at the time he did his original study.

b. The methodology was flawed in two arenas. Most importantly, a pulse oximeter was used to measure oxygen saturation in their exercising subjects. Unfortunately pulse oximeters should not be used to measure oxygen saturation during exercise as that technique can lead to falsely low readings.^(17,18) Additionally, no measures of ventilatory function were performed and there was no assessment of actual ventilatory and respiratory function in individuals in the restraint position;

c. The author’s reported a “statistically significant” difference between the two groups, however, they did not report the actual statistics that they used to reach this conclusion, and if one applies the proper statistics to this situation, no statistically significant difference exists and it can easily be seen that the difference between their groups was generated by just one “outlier.”

d. Finally, the authors noted that all the subjects returned to “normal” (albeit 20 seconds longer in the experimental group) thus it is hard to understand how one can conclude that the restraint position “causes” positional asphyxia as later pathologists have concluded. Ironically, Reay et al. suggested that additional work should be performed on this topic and, in 1997, my co-workers and I reported our experiments using a more appropriate approach and more sophisticated methodology than Reay, et al. We performed pulmonary function testing (forced vital capacity; forced expiratory volume in 1 second and maximal voluntary ventilation)

on fifteen individuals, with an age range of 18 to 40 years, in the sitting, supine, prone, and maximal restraint position (hog-tied). The subjects were then subjected to exercise (Exercise consisted of four minutes of heavy exercise on a bicycle ergometer; a calibrated bicycle exercise machine) and then the subjects were either restrained in a “hog-tied” position or allowed to rest in a sitting position. During the post-exercise periods (and when the subjects were either sitting or in a hog-tie position), determinations of arterial blood gas tensions, pulse rate, oxygen saturation and pulmonary function testing (PFT) were performed. Placing individuals in the restraint position after exercise resulted in a restrictive pulmonary function pattern as measured by PFTs, however, these PFT changes (while statistically significant) were not clinically relevant and indeed were still within “normal limits.” Based upon arterial PO₂ determinations, the oxygenation of blood increased with exercise, which is what one would expect and which was in keeping prior published work on exercise physiology^(14,15,16). Most importantly, there was no evidence of hypoxia in the restraint position after exercise and no evidence of hypercapnia either during exercise or in restraint. Accordingly, my co-workers and I concluded that:

a. There is no evidence that body position while in the “hog-tie” or “hobble” restraint position as a factor in and of itself causes hypoventilation or asphyxiation.

b. Factors other than body positioning are more important determinants for the sudden, unexpected deaths that occur in individuals who are placed in the restraint position. We acknowledged that individuals who are extremely obese, and have a body mass index (BMI) greater than 30 kg/m (Lewis’ BMI was 29) might be at a greater risk for developing further restrictive pulmonary function secondary to abdominal compression from body positioning, but that was speculative and eventually would receive further investigation^(11,12). Of note is that the current clinical evidence suggests that the prone position improves blood oxygenation compared to the supine position.^(19,20,21,22,23,24)

12. The same month our article was published, an editorial appeared in the British Medical Journal titled, “Acute Excited States and Sudden Death: Much Journalism, Little Evidence.”⁽²⁵⁾ The authors, Farnham and Kennedy, made a number of important points:

a. Excited delirium is commonly associated with cocaine and other stimulants, and less

1 commonly with mental illness.

2 b. Before neuroleptics were introduced, death in these cases was alluded to as “exhaustion.”

3 c. Acute excited states have had a variety of names but have a high mortality and should be
4 regarded as a medical emergency.

5 d. Death is preceded by a cycle of alternating struggle and collapse.

6 e. There is a lack of anatomical findings to explain death.

7 f. If a state of excited delirium cannot be prevented or the situation defused, and the
8 individual is a danger to themselves or others, the only other options are restraint, seclusion, or
9 medication.

10 Although this article was written before our article was published, the authors stated in regard to
11 the concept that death was due to positional and/or restraint asphyxia: “this suggestion must be
12 treated with caution.”⁽²⁵⁾ Farnham and Kennedy pointed to the source of the problem with deaths
13 due to excited delirium, as perceived by the legal system, the public, and the press: “Legal
14 reasoning favours single proximate causes rather than medical conditions, but the intervention
15 most proximate to the time of death is not necessarily the cause of death. Similarly, popular
16 journalism favours controversy and blame rather than balance and exploration.”⁽²⁵⁾

17 13. In 1998, Pollanen et al. reported 21 sudden unexpected deaths in association with
18 excited delirium.⁽²⁶⁾ All exhibited symptoms of excited delirium: bizarre or hyperactive behavior,
19 paranoia, shouting, thrashing about, and ranting. The most interesting aspect of this report is the
20 distribution of cases by etiology of the excited delirium. In 12 individuals (57%), the excited
21 delirium was due to psychiatric disorder; in 8 (38%) to cocaine, and in one a combination of
22 alcohol, morphine, diazepam, acetaminophen, and marijuana. The mean age was 33 years; 20
23 were males; all were restrained; and 18 were placed in a prone position. The report stated 3 had
24 pressure on the neck, and 8 of the eighteen restrained prone also had chest compression; all
25 suddenly lapsed into “tranquility” shortly after being restrained; and nineteen died at the time of
26 restraint. The other two were resuscitated but in a deep coma and died several days later. The
27 authors concluded that they “could not establish a definitive causal link between unexpected
28 death and restraint in people with excited delirium.”⁽²⁶⁾

14. The paper by Stratton et al. in 2001 ⁽²⁷⁾ is interesting and highly relevant in that all the reported twenty deaths due to excited delirium syndrome were witnessed by Emergency Medical Services (EMS) personnel who were able to institute immediate CPR. The study involved 216 cases of excited delirium witnessed by EMS personnel. In all cases, the individuals had been restrained in some sort of hobble restraint. Of the 216, 20 experienced cardiopulmonary arrest and died. Two deaths were excluded from the study, one because of pulmonary emboli and the other because of ligature marks and contusions of the neck. The presenting pattern for the 18 deaths were similar: (a.) excited delirium; (b.) hobble restraint; and (c) forceful struggle against restraint. Analysis of their data reveals that of the 18 individuals, 9 had heart weights that were above 2 standard deviations from the norm (and an enlarged heart is a major risk factor for sudden death) when the height of the individual was used to assess normal heart weight and another 2 had heart weights more than 1.5 standard deviations from the norm. ⁽²⁸⁾ As only 2.5% of a normal population will have a heart weight greater than 2 standard deviations from the norm, that 1/2 of this population was greater than 2 standard deviations from the norm, strongly suggests that underlying cardiovascular disease was over represented in this population. This is not surprising in light of the fact that 45% of this population is reported to have known chronic cocaine use. It is also of note that apparently no cardiac arrests occurred in which there were successful resuscitations. The combination of the presumed short interval between the occurrence of cardiac arrest and resuscitative measures, coupled with the observation that there were no successful resuscitations strongly suggests that asphyxia did not play a role in these deaths and that the pathology was predominantly cardiac. This low rate of resuscitation is in keeping with the nationally reported outcomes of cardiac arrests due to heart disease in large cities. Another interesting observation from this paper is that, from 1992-1996 patients were restrained in a hog-tie position. Subsequent to 1996 the less restrictive hobble (also called the TARP; total appendage restraint position) was used. The death rate while individuals were restrained in the hogtie position was 11%. After the less restrictive position was adopted the death rate remained 11% once more suggesting that the position itself was not a factor in the cause of death. All cardiopulmonary arrests were preceded by a short period (estimated at 5

1 minutes or less) during which the struggle had ceased and the individual had labored to shallow
2 breathing.

3 15. In order to continue to support the hypothesis that restraint causes low blood
4 oxygen levels (a hypothesis for which there exists no supportive experimental data) and hence
5 causes positional asphyxiation, some individuals now claim that these deaths are due to
6 compromise in ventilation occurring when an officer/medical worker applies body weight to the
7 upper torso of an individual in an attempt to restrain the individual and/or prevent further
8 struggle. This is usually accomplished by lying across an individual's back, or by applying
9 pressure on the back with a knee or hands. The first significant mention of this concept was by
10 O'Halloran and Frank in a paper published in 2000.⁽²⁹⁾ They also stated that they felt that the
11 term "restraint asphyxia" should be used in such cases rather than positional asphyxia.
12 Interestingly, the paper makes no reference to our work in this arena. Nevertheless, O'Halloran
13 and Frank report on twenty-one cases of "asphyxial death" during prone restraint. Eight had a
14 history of chronic mental illness. All were in a prone position. There was no use of chokeholds.
15 The lack of relationship to the hog-tie position is illustrated by the fact that only four of these
16 individuals were hog-tied; three of them had enlarged hearts (greater than two standard
17 deviations from normal heart weight in relation to body mass) and in the fourth, heart weight was
18 not reported. Toxicology revealed that one had a very high blood level of cocaine; another
19 methamphetamine and cocaine metabolite, and another methamphetamine. The fourth had a
20 three-year history of psychosis and a therapeutic level of haloperidol (an anti-psychotic drug in a
21 class of drugs that have been related to sudden deaths) in the blood. The most striking fact about
22 these cases is the fact that eleven had stimulants in their blood, eight individuals had a history of
23 chronic mental illness; three had heart disease. Of the remaining five, four were on medications
24 that are associated with induced prolongation of the QT interval (the time between the beginning
25 of the Q wave and the T wave on an electrocardiogram; in some cases this has been associated
26 with an increased risk of sudden death). Importantly, virtually all of the individuals who died had
27 pre-existing conditions that combined with hyperactivity were more than enough to explain the
28 death without invoking asphyxia as a mechanism. As has occurred so frequently before in the

1 past, the classic mistake of confusing proximity of an action, e.g., restraint, with causality (an
2 error in logic identified by Aristotle more than 2000 years ago)⁽³⁰⁾ was not considered as a
3 possibility.

4 16. In order to better assess the true relationship between weight force on the back and
5 its effect upon ventilation, my co-workers and I published the results of our work in this area in
6 2004 and 2005.^(31,32) We conducted a series of experiments in which weights were applied to
7 individuals restrained in the hog-tie or hobble position (i.e. maximal restraints). Previously,
8 proponents of the theory of asphyxia secondary to restraint, have suggested that most deaths
9 reported in restrained individuals involve two modalities: either the individual is held down in a
10 four-point restraint or the individual's hands are cuffed behind the back and the ankles tied
11 together. Thus, to simulate the "worst case" situation in our experiments design we utilized the
12 most severe form of restraint. We utilized three positions: sitting; hog-tie with 25 lb on the back,
13 and hog-tie with 50 lb on the back. We then measured oxygen saturation by pulse oximetry, end-
14 tidal CO₂ (carbon dioxide) levels, forced vital capacity (FVC), and forced expiratory volume in 1
15 second (FEV₁). FVC and FEV₁, while significantly lower in the restraint positions compared to
16 the sitting, were not significantly different with or without weight force. More importantly, the
17 mean oxygen saturation levels were above 95% and mean end-tidal CO₂ levels were below 45
18 millimeters of mercury for all positions.⁽³¹⁾ Thus, the hog-tie position, with or without 25 and 50
19 lb of weight force, while producing a restrictive pulmonary function pattern, did not produce any
20 evidence of hypoxia, hypoventilation, oxygen desaturation or hypercapnia. Accordingly, persons
21 with lesser forms of restraint applied like Mr. Lewis would be expected to likewise suffer no
22 clinically significant changes to blood oxygen levels.

23 Some persons also argue that if one puts more weight on the individual eventually
24 hypoxia will result. Obviously, if enough weight is placed upon an individual's chest, thereby
25 crushing or immobilizing it, the individual can asphyxiate. There is however, no proof that the
26 amount of force placed on individuals by kneeling on them or laying across their bodies'
27 compromises ventilation to the point that hypoxemia would occur. Although Mr. Lewis had two
28 broken ribs, one was likely the result of CPR efforts (rib 7), and the other would not inhibit his

ability to breathe (rib 9). “In fact, these activities are performed daily by police making arrests of violent individuals and medical personnel restraining violent individuals.”⁽³³⁾ In this case, Mr. Lewis merely had weight force briefly applied to his extremities with Officer Martinez and Mulhern applying weight force until Lewis returned to the ground. Additionally, now experiments with up to 225 pounds of weight placed upon individuals’ backs have been performed and they do not result in alterations of ventilation sufficient to cause clinically important effects upon blood oxygenation.⁽³²⁾ Once again there is no study in the literature that demonstrates weight applied to the back lowers blood oxygen levels.

17. To quote from Dr. Di Maio (Editor in Chief of the American Journal of Forensic Medicine and Pathology and author of the most widely modern textbook of Forensic Medicine from his most recent book titled Excited Delirium Syndrome⁽³⁴⁾:

“Acceptance of the concept of positional or restraint asphyxia as the cause of death often involves suspension of common sense and logical thinking. Originally, deaths in association with excited delirium syndrome and ascribed to positional asphyxia involved individuals either placed in a situation where respiration was impaired by a compressive force on the abdomen or tied up in a way alleged to restrict respiration, and thus oxygenation of blood, e.g., hog-tying. The former concept has some legitimacy. Thus, an individual restrained and placed in the back of a car such that the abdomen is over the transmission hump is probably a true example of positional asphyxia. Even in these cases, however, it was stated that drugs were usually present in such individuals and contributed to the death.

Almost immediately after the concept of positional asphyxia was offered, the concept was expanded such that whenever anyone is restrained and dies, positional or restraint asphyxia is said to be the cause of death whatever the position of the deceased, the method of restraint, or the presence of drugs. In spite of the work of Chan et al., which essentially disproved the concept of positional asphyxia proposed up to that time, many individuals still cling to this essentially discredited concept. This is not to say that positional asphyxia cannot be a cause of death in association with excited delirium syndrome. Rather, it is a rare occurrence usually involving unusual positioning of the individual, e.g. an obese individual, hog-tied and wedged between the front and backseat of a vehicle with the abdomen draped over the transmission hump. Rarely, deaths in association with excited delirium syndrome may be due to traumatic asphyxia. This occurs if a number of individuals lie or sit on an individual for several minutes, compressing the chest and abdomen, such that respiration is not possible. Whether an extremely obese individual, lying prone, handcuffed, and with bound feet has significant impairment in the ability to oxygenate blood is not clear. A number of factors would have to be considered, including the degree of obesity.

Chan et al conducted a series of experiments to determine if placing an individual prone in the hog-tied position, following strenuous exercise, produced restriction in ventilation such that there was impairment in oxygenation of blood. They found that while this resulted in restrictive pulmonary functioning as measured by pulmonary function tests (PFT), the changes were not clinically relevant. There was no evidence of hypoxia in the restraint position after exercise, as well as no evidence of hypercapnia either during exercise or in restraint.

In an attempt to counter Chan et al’s work and maintain the concept of positional/restraint

1 asphyxia, some investigators now claim that the death is due to compromise in ventilation
2 occurring when an officer or medical worker, attempting to restrain individuals, kneels on them
3 or lies across their backs in an attempt to prevent further struggle. Of course, as usual, no
4 scientific backing is given for this theory. Chan et al address this theory in a paper published in
5 2004. They conducted a study in which weights were applied to individuals restrained in the
6 hog-tied or hobble position, a position rarely used nowadays and the most extreme of the restraint
7 positions. The authors utilized three positions: sitting; hog-tied with 25 lb on the back, and hog-
8 tied with 50 lb on the back. They found that the hog-tied position, with or without 25 and 50 lb of
9 weight force, while producing a restrictive pulmonary function pattern, did not produce any
10 evidence of hypoxia or hypoventilation, i.e., no evidence of hypoxia, oxygen desaturation,
11 hypercapnia, or CO₂ retention. Thus, there is no proof that the force placed on individuals by
12 kneeling on them or lying across their bodies compromises respiration. In fact, these actions are
13 performed daily by police making arrests of violent individuals and medical personnel restraining
14 violent individuals without any untoward results.

15 18. Positional asphyxia may involve circumstances which arise out of massive obesity
16 and/or unusual positioning of the individual, however, this is still an area that demands
17 investigation and is merely a hypothesis. Too often in this arena, hypotheses have become
18 “truths”. Individuals who die while restrained do so as a result of multiple factors, however it is
19 now clearly been shown that the restraint position per se does not cause asphyxia in the usual
20 manner in which it is employed. Furthermore, arguments that perhaps drugs, weight, or any of a
21 number of other variables might somehow interact with restraint to cause asphyxiation do not
22 have logical validity. Drugs such as cocaine, methamphetamine and other stimulants as well as
23 PCP do not adversely affect breathing. Moderate obesity similarly does not adversely affect
24 breathing to a clinically relevant degree. In theory, someone might argue that our studies were
25 done in the daytime and these events took place at night, therefore our results do not apply. The
26 obvious response is that whether or not it is light outside does not affect breathing. The same is
27 true of stimulant drugs, whether weight is applied to the middle of the back or to the side, or
28 whether someone has or does not have underlying (non-pulmonary) illnesses. Thus, the critical
question is not whether restraint affects ventilation (it does) but rather how much does it affect
ventilation and how much ventilation is necessary to survive? In the setting of thoracic surgery,
when considering the likelihood that someone will survive a pulmonary resection for lung cancer,
most surgeons feel a post operative vital capacity of 25% of normal is required in order that the
individual does not live a “bed to chair” existence. Similarly, in the Emergency Department
people are generally not felt to be at major risk to life from an asthma attack until flow rates fall

1 to below about 20% of normal. Individuals with Guillian-Barré syndrome [a progressive process
2 characterized by loss of muscular strength that eventually can affect the ventilatory muscles.
3 Patients with botulism (a form of severe muscular paralysis most often caused by improper
4 canning of foods) are generally felt to be safe enough to breathe on their own, until ventilation
5 falls below 15 ml/kg (65 ml/kg is normal). Thus 20-25% of ventilatory function appears adequate
6 to maintain life as well as survive major chest surgery. Thus it therefore follows that if position
7 or weight on the back is the cause of asphyxial deaths, the weight applied should be great enough
8 to reduce ventilation below those levels. Furthermore, since it takes several minutes to
9 asphyxiate in the setting of no ventilation at all, when ventilation is reduced to some quantity
10 between 0% to 25% of normal levels, it will take increasingly longer to asphyxiate.

11 In this case, Mr. Lewis was placed in a prone position to facilitate the application of
12 handcuffs and the WRAP. Weight force was applied to Mr. Lewis for three to five seconds by
13 Officer Martinez, and fifteen to thirty seconds by Officer Mulhern, and terminated as soon as
14 Lewis went back to the ground. After Mr. Lewis was handcuffed, and the WRAP was applied to
15 his legs only, he was rolled over and placed in a seated position. As the Hayward Fire
16 Department approached the scene, he was laid back down on his back to facilitate treatment.

17 19. In conclusion, it is my opinion that Mr. Lewis' death associated with "excited
18 delirium" resulted from a fatal cardiac arrhythmia due to multiple factors (i.e. "excited delirium",
19 exercise, and underlying cardiovascular disease) all of which combined in a non-quantifiable way
20 to result in his death. While restraints in general increase the psychological and physiological
21 stress on the individual, there is no evidence that body position as a factor by itself causes
22 hypoventilation, respiratory compromise, or positional asphyxia in the hogtie or hobble custody
23 restraint position. The hypothesis that Mr. Lewis' position per se directly contributed to his
24 death through an asphyxial mechanism cannot be supported by experimental data and the data do
25 exist, essentially prove that such a mechanism could not have taken place. The arguments that it
26 was the weight applied to Mr. Lewis' back cannot be substantiated by the sworn testimony as the
27 only weight force applied to Lewis was temporary in nature and terminated as soon as Lewis
28 went back to the ground (the weight force applied 5 seconds by Martinez and 15 to 30 seconds by

1 Mulhern.) Furthermore such a brief period of time is insufficient to cause death by asphyxiation
2 and the injuries that Lewis suffered were not consistent with being crushed to death. (“negative
3 pathology”) Arguments that the “ecologic validity” (e.g. differing field conditions) of the work
4 that has been done thus far is not of a high enough level are without physiologic rationale and
5 make no more sense than (as mentioned above) criticizing the work because it did not take place
6 at night.

7 I declare under penalty of perjury under the laws of the United States that the foregoing is
8 true and correct, and that this declaration was executed on November 11/30, 2005 in San Diego,
9 California.

10
11 /s/
12 TOM NEUMAN, M.D.

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